

**Expert witness report:  
The rationale for proof of vaccination requirements  
in Chicago and Cook County, Illinois**

**This expert witness report was prepared by Andrew Bostom, MD, MS.**

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This report was prepared by *pro bono* Andrew Bostom, MD, MS, for *Ravago v. Lightfoot* in the U.S. District Court for the Northern District of Illinois on **February 16, 2022**.

Andrew Bostom, MD, MS

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Print Name

AGB

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Signature

**Cases in which Andrew Bostom, MD, MS, has offered expert testimony:**

Brian C. Dolata v. City of Deland — Case #: 5D20-1925 - Fifth District Court of Appeal

Brian C. Dolata v. City of Deland — Case #: 2020 10900 CIDL – Volusia

Gerald Carroll v. Gadsden County — Case # 20000542CAA – Gadsden

Southwell v. McKee — Rhode Island Superior Court

Zulay Rodriguez-Velez v. Pedro R. Pierluisi-Urrutia — Puerto Rico, Federal Court

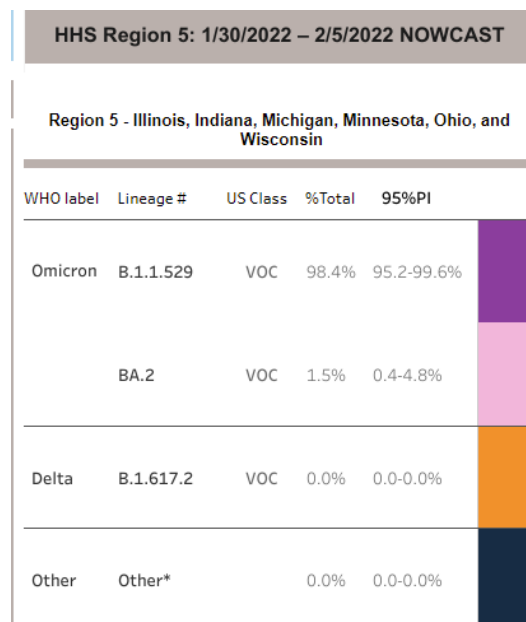
Tropical Chill Corp et al v. Pierluisi-Urrutia et al — Puerto Rico, Federal Court

**Attached to the expert witness report document:**

Curriculum vitae including publications for Andrew Bostom, MD, MS

**1) Omicron is dominant in Illinois & Chicago.**

The Centers for Disease Control and Prevention (CDC) SARS-CoV-2 variant surveillance data through February 5, 2022 indicate that omicron B.1.1.529 and BA.2 account for essentially all of the virus circulating in the Illinois (and neighboring states) area.<sup>1</sup>

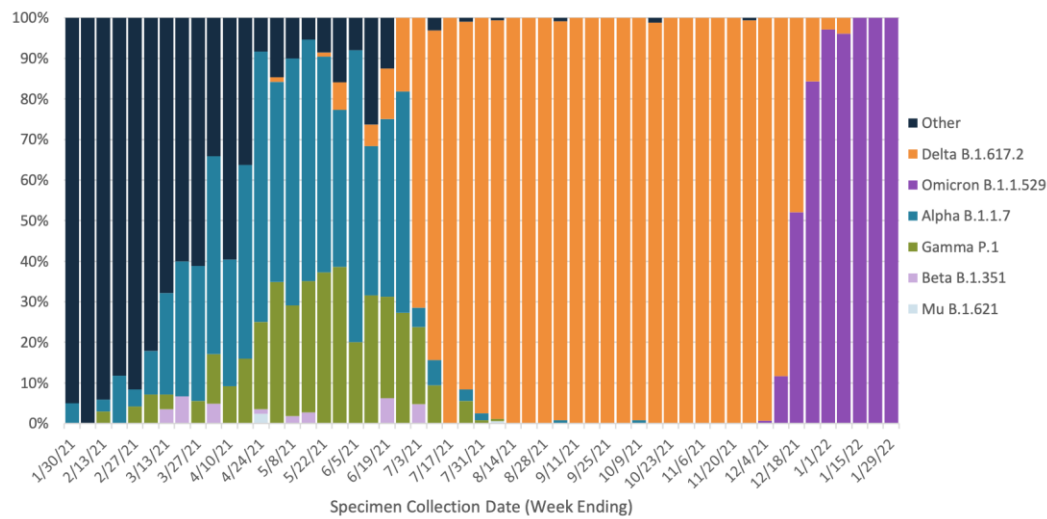


The same is true in Chicago. The latest report from the Chicago Department of Public Health shows Omicron comprising 100% of SARS-CoV-2 variant lineage proportions.<sup>2</sup>

<sup>1</sup> Centers For Disease Control and Prevention COVID Data Tracker—Variant Proportions <https://covid.cdc.gov/covid-data-tracker/#variant-proportions>; <https://covid.cdc.gov/covid-data-tracker/#nowcasting>

<sup>2</sup> [https://www.chicago.gov/content/dam/city/sites/covid/reports/2022/CDPH\\_GISAID\\_Proportions\\_2-10-22.pdf](https://www.chicago.gov/content/dam/city/sites/covid/reports/2022/CDPH_GISAID_Proportions_2-10-22.pdf)

### SARS-CoV-2 Lineage Proportions in Chicago (GISAID)\*



Likewise, suburban Cook County data show Omicron comprised 91% of variants detected in late January/early February 2022.

COVID-19 Variants Detected All Time and Prior Two Weeks Suburban Cook County Health Department

Variant	Total	Percent of Total	Count Last Two Weeks	Percent Last Two Weeks	Percent Change from Prior Two Week Period
Delta	4671	60.2%	13	8.1%	-50.4%
Alpha	1352	17.4%	0	0.0%	0.0%
Omicron	1020	13.1%	146	91.2%	+11.7%
Gamma	454	5.9%	0	0.0%	0.0%
Other	228	2.9%	1	0.6%	-67.9%
Beta	29	0.4%	0	0.0%	0.0%
Mu	6	0.1%	0	0.0%	0.0%
Lambda	0	0.0%	0	0.0%	0.0%

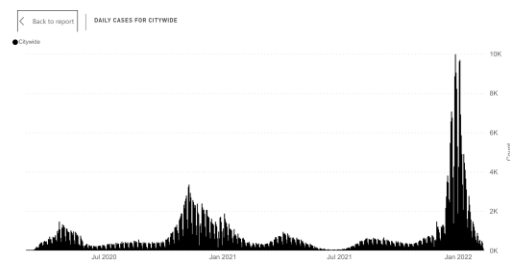
*Table includes all variants currently designated by the World Health Organization as a Variant of Concern or Variant of Interest. SARS-CoV-2 variants are identified through genomic sequencing. Only a small proportion of laboratory specimens positive for SARS-CoV-2 are sequenced. Some of these specimens are selected through random sampling and others are sequenced as a part of public health investigations, or through provider ordering. Therefore, the proportions represented here may not be representative of all variants circulating in suburban Cook County. The majority of these specimens were collected after February, 2021. Percent change is calculated on variant proportions, rather than counts, to account for variable sampling from week to week. These data are updated weekly on Wednesdays.*

## 2) Vaccination does not stop Omicron variant transmission.

Covid-19 case numbers in Chicago, suburban Cook County, and Illinois were at all-time highs during the Omicron surge, despite high vaccination rates<sup>3</sup>.

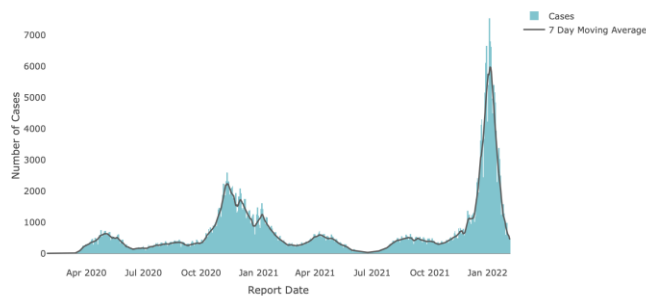
### COVID Dashboard

Data are updated M-F at 5:30 p.m., except for City holidays



Chicago<sup>4</sup>

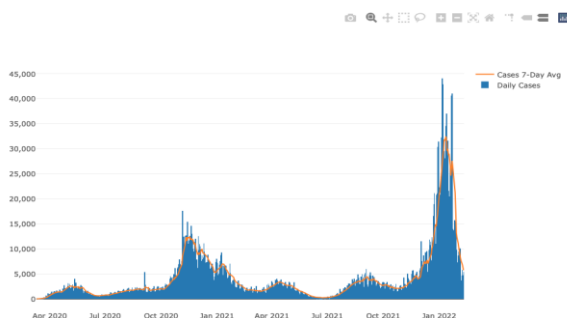
COVID-19 Cases by Report Date in Suburban Cook County, IL  
(n = 495,064)



Suburban Cook<sup>5</sup>

### Cases Change Over Time

Illinois



<sup>3</sup> As of February 12, 2022, 71% of Illinois residents age 5+ are fully vaccinated, as are 81% of Chicago residents and 70% of Cook County residents (Chicago included)

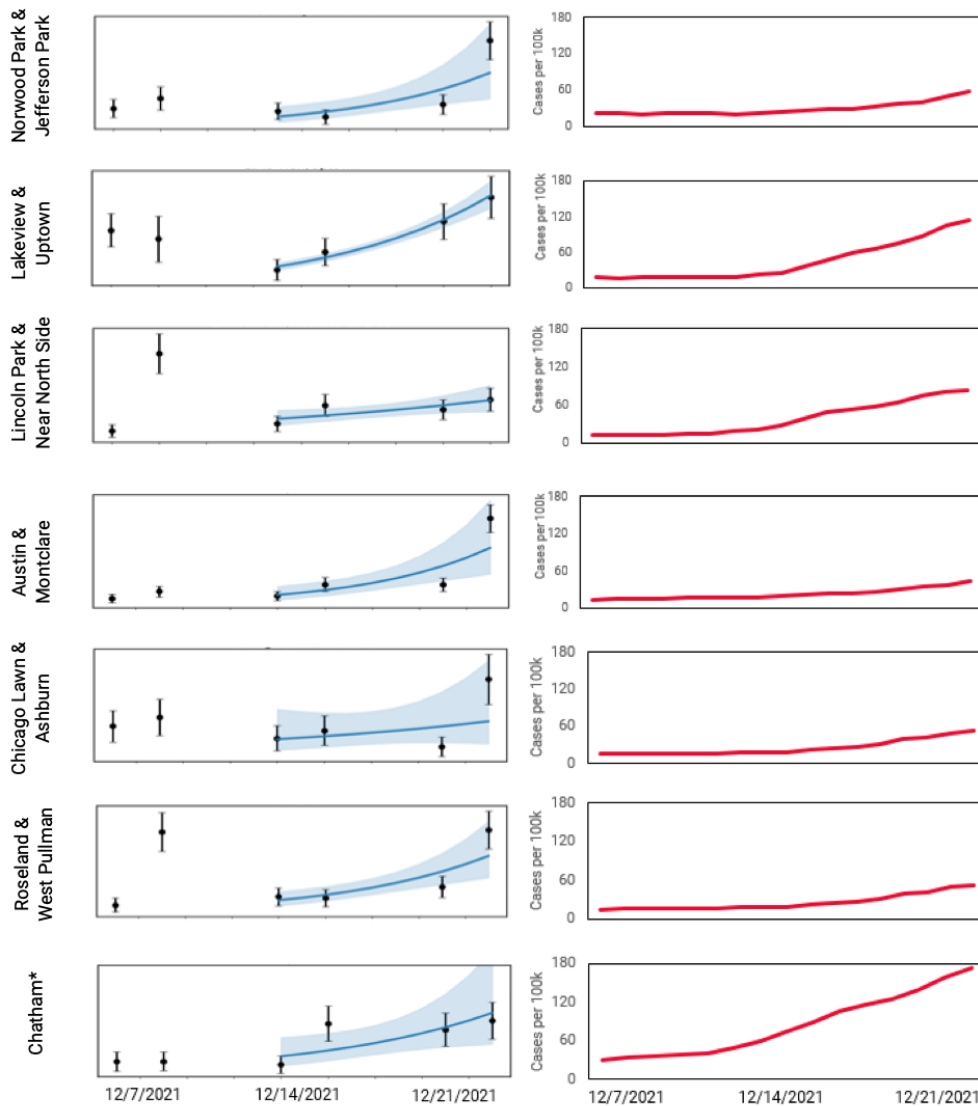
<sup>4</sup> <https://www.chicago.gov/city/en/sites/covid-19/home/covid-dashboard.html>

<sup>5</sup> <https://ccdphcd.shinyapps.io/covid19/>

**State of Illinois<sup>6</sup>**

Additionally, though the data are limited, Chicago Department of Public Health wastewater surveillance<sup>7</sup> show higher SARS-CoV-2 prevalence in catchment areas with higher vaccination rates.

**Figure 4: Amount of SARS-CoV-2 in wastewater (left) and reported cases of COVID-19 (right) for each catchment zone in CDPH's wastewater monitoring program – Chicago, Illinois, December 2021.** Y-axis values for wastewater data are not provided because the values have not been corrected to account for differences in population or other factors between catchment zones. The blue line represents the estimated two-week trend. The blue shaded area is the 67% confidence interval for this trend line. Reported cases of COVID-19 are shown as 7-day rolling averages (in red). For large catchment zones, the COVID-19 case data covers the same population as the wastewater data. For the Chatham catchment zone, which has a small population, COVID-19 case data for the entire ZIP code is shown.



<sup>6</sup> <https://dph.illinois.gov/covid19/data.html>

<sup>7</sup> [https://www.chicago.gov/content/dam/city/sites/covid/documents/wastewater/Dec\\_CDPH\\_Wastewater\\_Report\\_19Jan2022.pdf](https://www.chicago.gov/content/dam/city/sites/covid/documents/wastewater/Dec_CDPH_Wastewater_Report_19Jan2022.pdf)

### 3) Omicron reduces the likelihood of being hospitalized for Covid-19.

A recent analysis from the South African government's National Institute for Communicable Diseases provides reason for optimism: S-Genes Target Failure (i.e., presumptive Omicron) cases are 80% less likely to be hospitalized.

**Table 1.** Multivariable logistic regression analysis evaluating the association between S gene target failure (SGTF) infection, compared to non-SGTF infection, and hospitalisation, South Africa, 1 October – 30 November 2021<sup>a</sup> (N=11,255)

	Hospital admission <sup>b</sup> n/N (%)	Adjusted odds ratio (95% CI)	P-value
<b>SARS-CoV-2 variant</b>	N=11,495		
SGTF	256/10,547 (2)	0.2 (0.1-0.3)	<0.001
Non-SGTF	121/948 (13)	Ref	-

<https://www.medrxiv.org/con-tent/10.1101/2021.12.21.21268116v1.full.pdf>

Comparable data from Scotland also yielded the same optimistic conclusion: “Early national data suggest that Omicron is associated with a two-thirds reduction in the risk of COVID-19 hospitalization when compared to Delta.”<sup>8</sup>

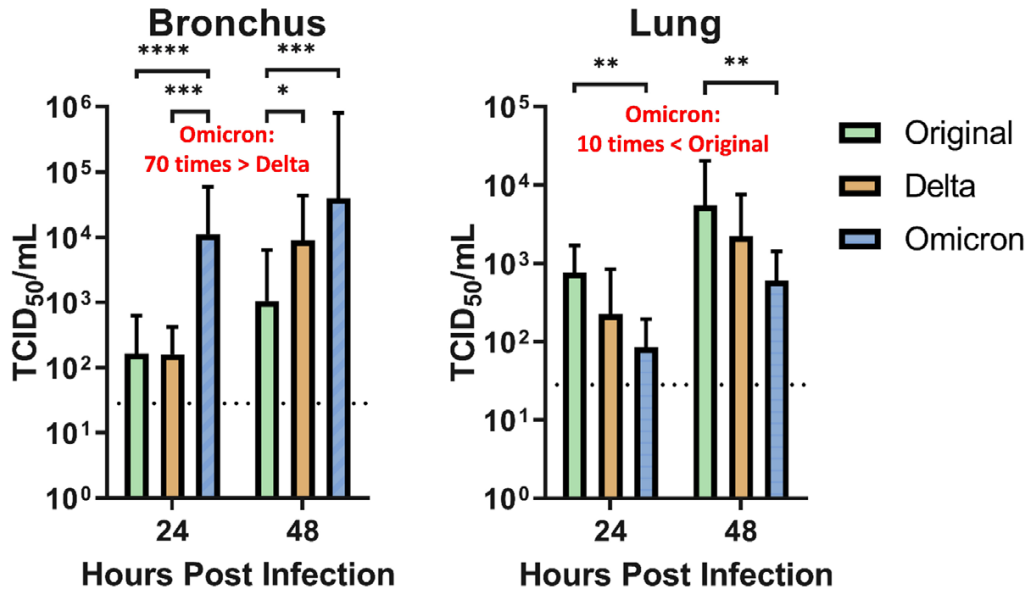
Denmark’s data reveal Omicron cases were three times less likely to end up with hospital admissions than the previous dominant variant, Delta.<sup>9</sup>

Hong Kong University researchers pointed to the likely reason, or mechanism, for Omicron’s increased infectiousness but reduced virulence: it replicates far more efficiently in the bronchus and upper respiratory tract than Delta, but less efficiently in the lungs.<sup>10</sup>

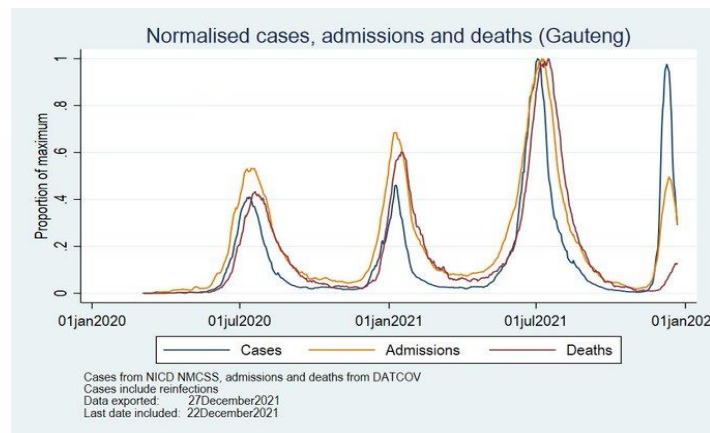
<sup>8</sup> <https://www.research.ed.ac.uk/en/publications/severity-of-omi-cron-variant-of-concern-and-vaccine-effectiveness->

<sup>9</sup> <https://arstechnica.com/science/2021/12/omicron-cases-less-likely-to-require-hospital-treatment-studies-show/>

<sup>10</sup> <http://www.med.hku.hk/en/news/press/20211215-omicron-sars-cov-2-infection>



But the most compelling evidence of Omicron ending any grave danger from SARS-CoV2 comes from South Africa, particularly the Gauteng province (population 18 million) where the first recognized Omicron wave occurred. According to Dr. Harry Moultrie of the South African government's National Institute for Communicable Diseases, Gauteng cases peaked on December 9 at 97 percent of the delta wave. Even more reassuringly, deaths were only 13 percent of the Delta peak.<sup>11</sup>



A recently-published working paper by a South African team of scientists who were conducting a sero-epidemiological survey in the Gautang Province confirms the

<sup>11</sup> <https://twitter.com/hivepi/status/1475383429403484163>

conclusion that Omicron infection is substantially less likely to require hospitalization or induce mortality than infection with other strains.

While cases may rise sharply as a wave of Omicron sweeps through a region, hospitalizations and deaths do not follow. The authors conclude: “We demonstrate widespread underlying SARS-CoV-2 seropositivity in Gauteng Province prior to the current Omicron-dominant wave, with epidemiological data showing an uncoupling of hospitalization and death rates from infection rate during Omicron circulation.”<sup>12</sup>

Based on their Omicron experience, some South African scientists have effectively declared the pandemic over, stating: “All indicators suggest the country may have passed the peak of the fourth wave at a national level... While the Omicron variant is highly transmissible, there has been lower rates of hospitalization than in previous waves. This means that the country has a spare capacity for admission of patients even for routine health services.”<sup>13</sup>

Thus, the first country to experience an Omicron wave has unambiguously concluded that the dominant variant presents no grave danger.

Initial U.S. data were available in a preprint from a team at Case Western Reserve University, which used propensity matched-cohort analysis to find markedly reduced disease severity during the period from December 14 to December 24, 2021. On an age- and risk-matched basis, they found ER visits were 70% lower than earlier cohorts, hospitalizations were 56% lower, ICU admissions were 67% lower, and ventilation were 84% lower.<sup>14</sup>

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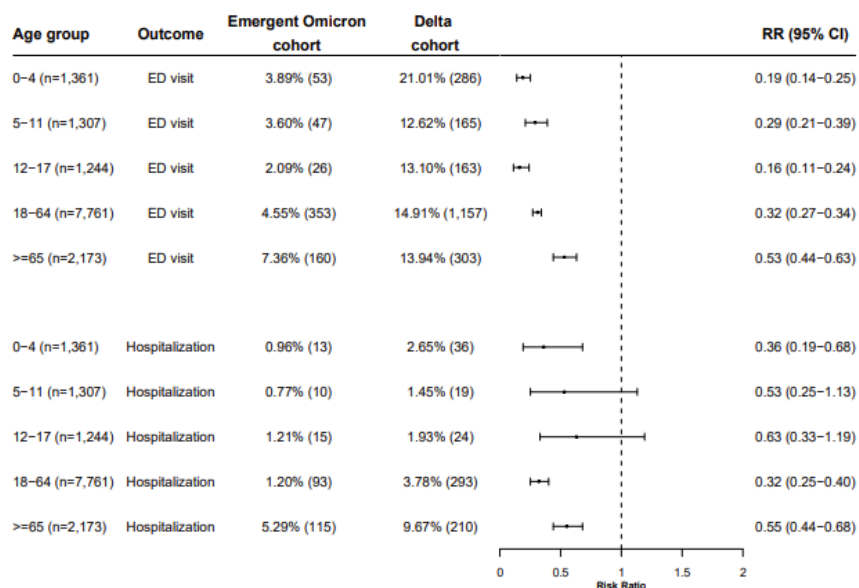
<sup>12</sup> <https://www.medrxiv.org/content/10.1101/2021.12.20.21268096v1>

<sup>13</sup> <https://sacoronavirus.co.za/2021/12/30/media-release-cabinet-approves-changes-to-covid-19-regulations/>

<sup>14</sup> <https://www.medrxiv.org/content/10.1101/2021.12.30.21268495v1>



**Age-stratified comparison of 3-day acute outcomes  
in matched patients with SARS-CoV-2 infections  
Emergent Omicron cohort (12/15–12/24) vs. Delta cohort (9/1–11/15)**



<sup>1</sup> <https://www.medrxiv.org/content/10.1101/2021.12.30.21268495v1>

As good as they appear, these reductions substantially *understate* the reduction of risk represented by Omicron, because this cohort included a non-negligible number of Delta infections. According to the authors: “The estimated prevalence of the Omicron variant during 12/15–12/24 was only 22.5–58.6%, suggesting that the outcomes for the Omicron variant may be found to be even milder than what we report here as the prevalence of the Omicron variant increases.”

Subsequently, these U.S. data were externally validated in a Kaiser-Permanente analysis which included 52,297 cases with SGTF (single gene target failure on PCR, a surrogate for Omicron) and 16,982 cases with non-SGTF (Delta [B.1.617.2]) infections, respectively.<sup>15</sup>

Per the authors:

*“Hospital admissions occurred among 235 (0.5%) and 222 (1.3%) of cases with Omicron and Delta variant infections, respectively. Among cases first tested in outpatient settings, the adjusted hazard ratios for any subsequent hospital admission and symptomatic hospital admission associated with Omicron variant infection were 0.48 (0.36–0.64) and*

<sup>15</sup> <https://doi.org/10.1101/2022.01.11.22269045>

0.47 (0.35-0.62), respectively. Rates of ICU admission and mortality after an outpatient positive test were 0.26 (0.10-0.73) and 0.09 (0.01-0.75) fold as high among cases with Omicron variant infection as compared to cases with Delta variant infection. Zero cases with Omicron variant infection received mechanical ventilation, as compared to 11 cases with Delta variant infections throughout the period of follow-up (two-sided  $p < 0.001$ ). Median duration of hospital stay was 3.4 (2.8-4.1) days shorter for hospitalized cases with Omicron variant infections as compared to hospitalized patients with Delta variant infections, reflecting a 69.6% (64.0-74.5%) reduction in hospital length of stay.”

**Table 1: Association of SGTF with adverse clinical outcomes.**

Outcome	Case population <sup>1</sup>	Cumulative events over observed follow-up (n of events per 1000 cases)		Event rate per 1000 person-days observed (n of events/person-days at risk)		Hazard ratio (95% CI)	
		No SGTF	SGTF	No SGTF	SGTF	Unadjusted	Adjusted
Any hospital admission	Cases tested in outpatient settings	189 (11.2)	88 (1.7)	0.71 (189/264,408)	0.30 (88/288,534)	0.32 (0.25, 0.42)	0.48 (0.36, 0.64)
	All cases	222 (13.1)	235 (4.5)	0.84 (222/264,424)	0.81 (235/288,608)	0.57 (0.47, 0.69)	0.72 (0.58, 0.88)
Symptomatic hospital admission	Cases tested in outpatient settings	187 (11.0)	84 (1.6)	0.71 (187/264,408)	0.29 (84/288,534)	0.31 (0.24, 0.41)	0.47 (0.35, 0.62)
	All cases	216 (12.7)	182 (3.5)	0.82 (216/264,424)	0.63 (182/288,608)	0.47 (0.38, 0.58)	0.62 (0.49, 0.77)
ICU admission <sup>2</sup>	Cases tested in outpatient settings	23 (1.4)	5 (0.1)	0.09 (23/266,778)	0.02 (5/288,694)	0.26 (0.10, 0.73)	--
	All cases	23 (1.4)	7 (0.1)	0.09 (23/267,378)	0.02 (7/289,617)	0.34 (0.14, 0.84)	--
Mechanical ventilation <sup>3</sup>	Cases tested in outpatient settings	11 (0.6)	0 (0.0)	0.04 (11/266,880)	0.00 (0/288,691)	--	--
	All cases	11 (0.6)	0 (0.0)	0.04 (11/267,480)	0.00 (0/289,631)	--	--
Death <sup>2</sup>	Cases tested in outpatient settings	14 (0.8)	1 (<0.1)	0.05 (14/266,844)	<0.01 (1/288,688)	0.09 (0.01, 0.75)	--
	All cases	14 (0.8)	1 (<0.1)	0.05 (14/267,444)	<0.01 (1/289,628)	0.09 (0.01, 0.75)	--

SGTF: S gene target failure, here interpreted as a proxy for SARS-CoV-2 Omicron variant infection (vs. Delta variant infection with non-SGTF samples); CI: confidence interval

<sup>1</sup>Sample sizes include 52,297 and 16,982 cases with and without SGTF, respectively, among whom 52,133 and 16,929 were tested in outpatient settings, respectively. We define symptomatic hospital admissions as those occurring among cases with respiratory symptom onset dates on or  $\leq 14$  days before the date of admission.

<sup>2</sup>Adjusted hazard ratios were not estimated due to limited observations within covariate strata.

<sup>3</sup>Unadjusted and adjusted hazard ratios were not estimated due to the absence of SGTF infections resulting in ventilation. The probabilities ( $p$ -values) of observing zero ventilated patients among all SGTF patients and among SGTF patients first tested in outpatient settings, under the null hypothesis of equal daily likelihood of ventilation among patients with and without SGTF, are equal to  $6.7 \times 10^{-6}$  and  $6.8 \times 10^{-6}$ , respectively.

Even the highest Covid-19 risk patients, i.e., long-term care facility (LTCF) residents, appear to fare substantially better following SARS-CoV-2 omicron infection, relative to infection with pre-omicron SARS-CoV-2 strains based upon preliminary data from the U.K. Among  $n=1,241$  LTCF residents infected during the omicron period, multivariable adjusted hospitalization rates were 50% lower compared to 398 residents in the pre-omicron period, while covid-19 related deaths were >70% lower.

Adding to the lack of any grave danger, there is also strong early evidence that Omicron infection offers robust protection against the Delta variant. This means that even if the Delta variant still presented a grave danger, it would be *counterproductive* to stop or slow the spreading of the presently dominant Omicron variant. A laboratory study at the Africa Health Research Institute<sup>16</sup> found:

“Importantly, there was an enhancement of Delta virus neutralization, which increased 4.4-fold. The increase in Delta variant neutralization in individuals infected with omicron

<sup>16</sup> [https://www.ahri.org/wp-content/uploads/2021/12/MEDRXIV-2021-268439v1-Sigal\\_corr.pdf](https://www.ahri.org/wp-content/uploads/2021/12/MEDRXIV-2021-268439v1-Sigal_corr.pdf)

may result in decreased ability of Delta to re-infect those individuals. Along with emerging data indicating that Omicron, at this time in the pandemic, is less pathogenic than Delta, such an outcome may have positive implications in terms of decreasing the Covid-19 burden of severe disease.”

Finally, at present, the Covid-19 infection fatality rate, IFR=0.06%<sup>17</sup>, is below that for seasonal flu, i.e., 2017-18 flu rates per the U.S. CDC<sup>18</sup>, IFR=0.13%.

#### **4) Omicron has not burdened the Chicago, Cook County, or Illinois hospital systems.**

IDPH, Suburban Cook DPH, and CDPH maintain data on the number of patients who are hospitalized for any reason and test positive for Covid upon admission or during their stay, but not data on the number of patients hospitalized because of Covid. However, the Chicago, Cook County, and Illinois hospital systems do not show evidence of being highly stressed during the Omicron surge.

##### ***Chicago***

In Chicago, total Emergency Department visits<sup>19</sup> decreased by 40% in March 2020 and have not recovered to pre-pandemic levels. In two years, COVID-positive visits have not been a driver of rises in ED visits.

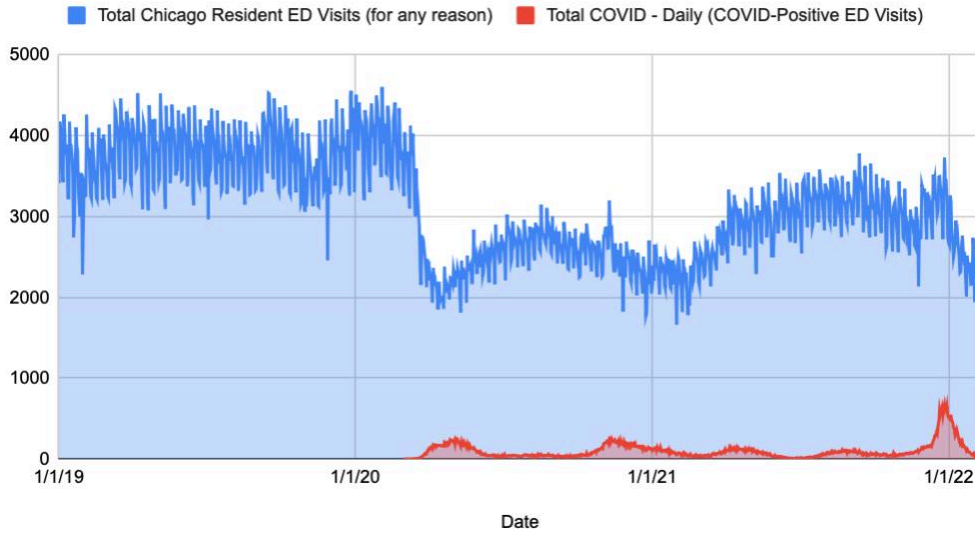
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<sup>17</sup> <https://www.telegraph.co.uk/news/2022/02/01/covid-really-deadly-flu-omicron-came-along/>

<sup>18</sup> <https://www.cdc.gov/flu/about/burden/2017-2018.htm#table1>

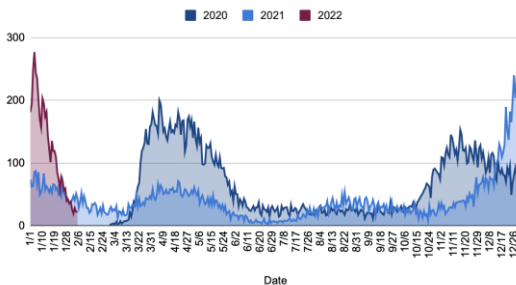
<sup>19</sup> <https://data.cityofchicago.org/Health-Human-Services/COVID-Like-Illness-CLI-and-COVID-19-Diagnosis-Emer/qwib-edaw>

### Chicago Hospital Daily ED Visits: 1/1/2019 - 1/19/2022

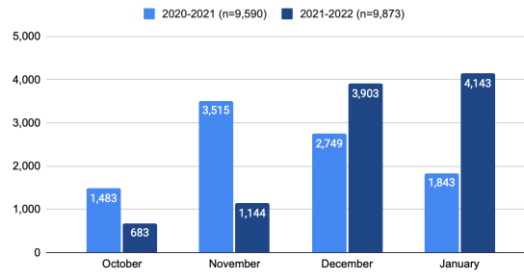


The peak of Covid-positive patients in the winter 2021-2022 Omicron surge has been comparable to fall/winter 2020<sup>20</sup>.

### Chicago Daily Covid-Pos Hospitalizations: 2020 - 2022



### Chicago Total Number of Monthly Covid Hospitalizations: Respiratory Virus Season Month Comparison

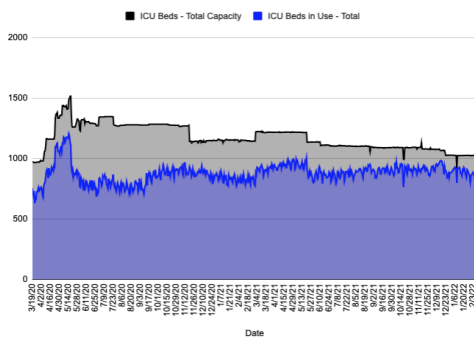


Chicago hospitals<sup>21</sup> did not see increases in total bed use during the Omicron peak. In fact, the number of available beds appears to be slightly higher than they were before the city increased surge capacity in the midst of the spring 2020 shutdown.

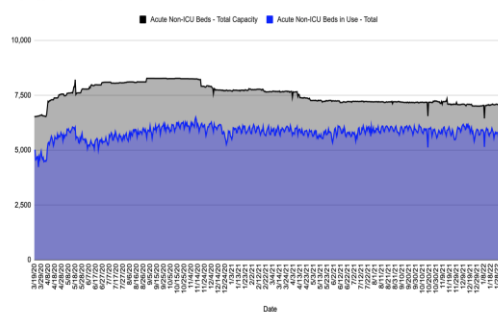
<sup>20</sup> <https://data.cityofchicago.org/Health-Human-Services/Monthly-Chicago-COVID-19-Cases-Deaths-and-Hospital/e9sn-zekj>

<sup>21</sup> <https://data.cityofchicago.org/Health-Human-Services/COVID-19-Hospital-Capacity-Metrics/f3he-c6sv>

Chicago ICU Beds: Total Capacity vs Use: 3/19/2020 - 2/10/2022

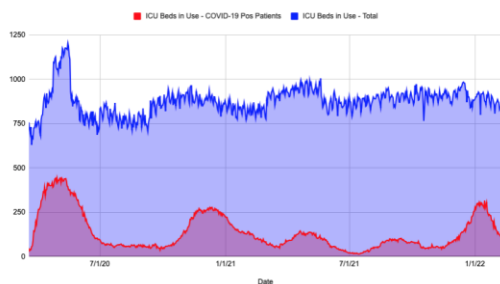


Chicago Acute Non-ICU Bed Capacity vs. Use: 3/19/2020 - 2/10/2022

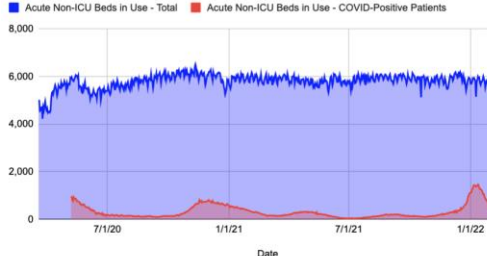


ICU and Acute Bed patient census indicates that, although the number of Covid-positive patients “comes and goes” in waves, it has little impact on total census. This, coupled with data that show Covid-positive patient census versus Non-Covid patient and Covid PUI<sup>22</sup>, strongly suggest many “Covid hospitalizations” are patients who are in the hospital for other reasons and test positive for SARS-CoV2 incidentally.

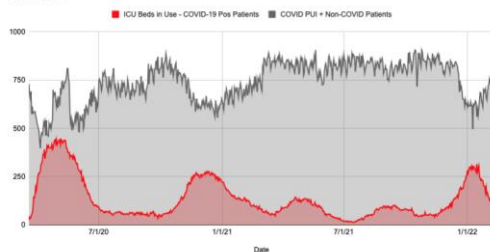
Chicago Total ICU Beds in Use vs. In-Use by COVID-Positive Patients: 3/19/2020 - 2/10/2022



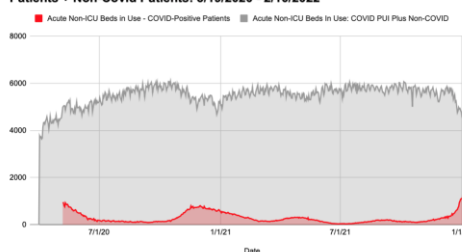
Chicago Total Non-ICU Beds in Use vs. In-Use by COVID-Positive Patients: 3/19/2020 - 2/10/2022



Chicago ICU Beds in Use: Covid Positive vs Covid PUI & Non-Covid, 3/19/2020 - 2/10/2022



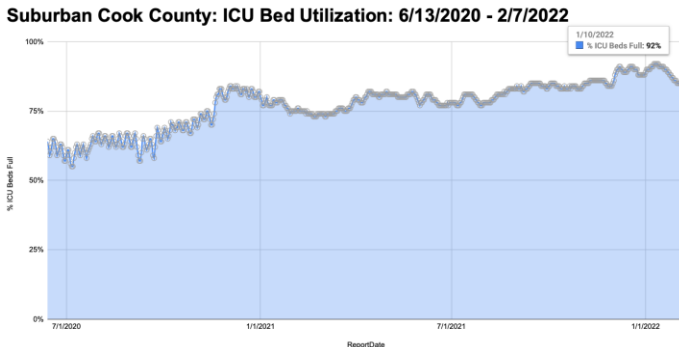
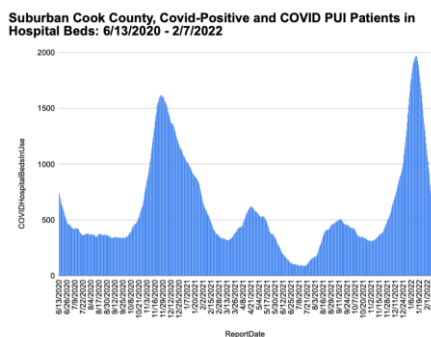
Chicago Non-Acute ICU Beds in Use: Covid-Positive Patients v Covid PUI Patients + Non-Covid Patients: 3/19/2020 - 2/10/2022



### Suburban Cook County

The peak number of Covid-positive and Covid PUI patients in suburban Cook County hospital during the Omicron surge exceeded the 2020 fall/winter wave. Overall ICU bed utilization peaked at 92%. Without data from spring 2020 – or from previous flu seasons – context is limited. It’s reasonable to expect that the patterns in the Chicago data hold for suburban Cook County.

<sup>22</sup> Person Under Investigation, a.k.a., awaiting a test result/diagnosis



**Illinois**

The trajectory of Illinois’ hospital-bed utilization<sup>23</sup> is similar to Chicago’s, likely because Chicago’s hospitals and Chicago’s increase in surge capacity, comprise a large portion of the data. February 2022 total beds appear to be at the same level as early April 2020.

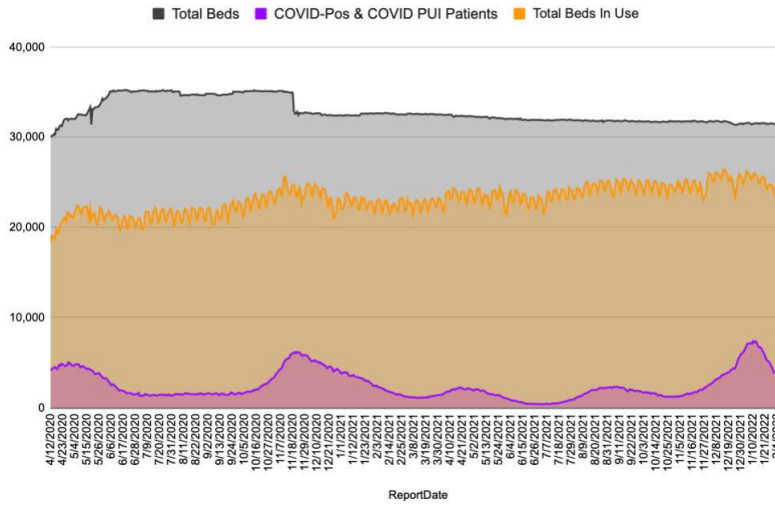
IDPH data<sup>24</sup> do not disaggregate Covid-positive patients from PUI patients, but HHS data for Illinois indicate PUI is anywhere from 5-10% of the total “Covid Patient” number, on average.

Similar to Chicago, bed use in Illinois hospitals does not appear to increase demonstrably with the number of Covid Patients.

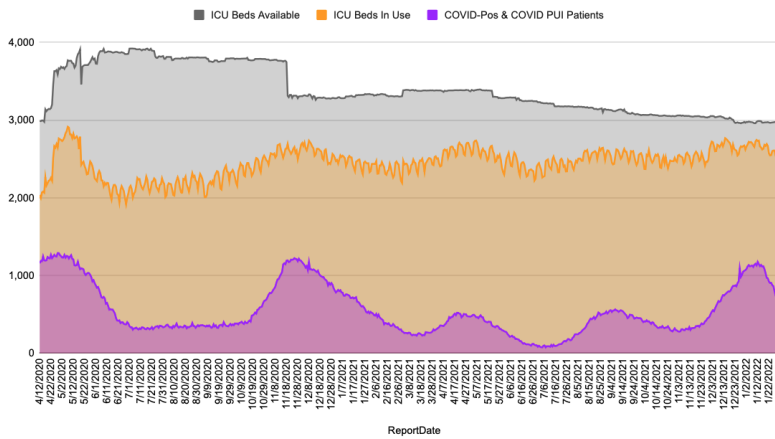
<sup>23</sup> <https://dph.illinois.gov/covid19/data/data-portal/covid-19-hospitalization-utilization.html>

<sup>24</sup> <https://dph.illinois.gov/covid19/data/data-portal/test-positivity-hospital-availability-cli-admissions.html>

### Illinois Inpatient Bed Utilization: 4/12/2020 - 2/9/2022



### Illinois ICU Bed Utilization: 4/12/2020 - 2/9/2022



In summary, Covid-19 vaccination cannot and has not stopped transmission of SARS-CoV-2, nor has it prevented the Omicron variant of SARS-CoV-2 from becoming dominant and prevalent in Illinois, Cook County, & Chicago. Multiple studies indicate that the risk of hospitalization and death from Omicron infection is dramatically reduced from previous variants. There is no evidence that the state’s, county’s, and city’s hospital systems have been stressed during the Omicron wave beyond levels experienced in previous respiratory virus seasons.

October, 2021

**CURRICULUM VITAE**  
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**EDUCATION**

1982	SUNY Health Science Center at Brooklyn (HSCB) College of Health Related Professions, Physical Therapy	B.S.
1983-1986	City University of New York (CUNY), Queens College Graduate School	M.S. Exercise Physiology
1986-1990	SUNY Health Science Center at Brooklyn (HSCB)	M.D. Cum Laude with Distinction in Research
1992-1994	Brown University Graduate School	M.S. Epidemiology (awarded 1999)

**POSTGRADUATE TRAINING**

1990-1992	Rhode Island Hospital, Providence, RI	Resident in Internal Medicine
1992-1995	Cardiovascular Disease Epidemiology National Heart, Lung, and Blood Institute/Public Health Service Framingham, MA Field Site	Clinical Investigator Pathway Fellowship in Cardiovascular Disease Epidemiology
1995-1997	Jean Mayer USDA Human Nutrition Research Center On Aging	Fellowship in Clinical Nutrition

**PROFESSIONAL LICENSES AND BOARD CERTIFICATION**

1982	Physical Therapy
1993	American Board of Internal Medicine
1997	Rhode Island Board of Registration in Medicine
2004	American Board of Internal Medicine Recertification
2012	National Lipid Association, Clinical Lipid Specialist
2014	American Board of Internal Medicine Recertification

**ACADEMIC APPOINTMENTS**

1995-2001	Scientist III, Jean Mayer USDA-HNRCA
1997-June 2001	Assistant Professor of Medicine, Brown University School of Medicine
July 2001- 2017	Associate Professor of Medicine, Brown University School of Medicine
Jul 2017-Jun 2021	Associate Professor of Family Medicine, Brown University School of Medicine



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### HOSPITAL APPOINTMENTS

1997-2001	Co-Director, Cardiac Rehabilitation Program, Memorial Hospital of Rhode Island
2001-2012	Director, Lipid Disorders Program, Division of Renal Diseases and Hypertension, Rhode Island Hospital
<b>2013-now</b>	Research Physician, Center for Primary Care and Prevention, Memorial Hospital of Rhode Island
2015-2017	Research Physician, Division of Renal Diseases and Hypertensions, Rhode Island Hospital

### MEMBERSHIPS IN SOCIETIES

1995-Present	American Heart Association - Council on Cardiovascular Disease, Epidemiology, and Prevention; Council on Arteriosclerosis
1997-2012	American Society of Nephrology
2010-Present	National Lipid Association

### PUBLICATIONS (Peer reviewed)

1. **Bostom AG**, Bates E, Mazzarella N, Block E, Adler J. Ergometer modification for combined arm-leg use by lower extremity amputees in cardiovascular testing and training. *Arch Phys Med Rehabil* 1987;68:244-7.
2. King ML, Guarracini M, Lennihan L, Freeman D, Gagas B, **Bostom AG**, Bates E, Nori S. Adaptive exercise testing for patients with hemiparesis. *J Cardiopulmonary Rehabil* 1989;9:237-42.
3. **Bostom AG**, Toner MM, McArdle WD, Montelione T, Brown CD, Stein RA. Lipid and lipoprotein profiles relate to peak aerobic power in spinal cord injured men. *Med Sci Sports Exerc* 1991;23:409-14.
4. **Bostom AG**, Eaton CB, Yanek L, McQuade W, Catalfamo J, Selhub J. Elevations in total plasma homocysteine in premature coronary artery, cerebrovascular, and peripheral vascular disease. *Atherosclerosis* 1993;102:121-4.
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9. Selhub J, Jacques PF, **Bostom AG**, D'Agostino RB, Wilson PWF, Belanger AJ, O'Leary DH, Wolf PA, Schaefer EJ, Rosenberg IH. Association between plasma homocysteine concentrations and extracranial carotid artery stenosis. *N Engl J Med* 1995;332:286-91.
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12. **Bostom AG**, Hume AL, Eaton CB, Yanek LR, Regan MS, Laurino JP, Craig WY, Perrone G, Jacques PF. The effect of high dose ascorbate supplementation on plasma lipoprotein (a) levels in patients with premature coronary heart disease. *Pharmacotherapy* 1995;15:458-64.
13. **Bostom AG**, Selhub J, Jacques PF, Nadeau MR, Williams RR, Ellison RC. Post-methionine load hyperhomocysteinemia in persons with normal fasting total plasma homocysteine: initial results from the NHLBI Family Heart Study. *Atherosclerosis* 1995;116:157-61.
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  105. Takahashi A, Hu SL, **Bostom A**. Physical Activity in Kidney Transplant Recipients: A Review. *Am J Kidney Dis.* 2018 Sep;72(3):433-443. doi: 10.1053/j.ajkd.2017.12.005. Epub 2018 Feb 23. PMID: 29482935 Review.
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  107. **Bostom A**, Pasch A, Madsen T, Roberts MB, Franceschini N, Steubl D, Garimella PS, Ix JH, Tuttle KR, Ivanova A, Shireman T, Gohh R, Merhi B, Jarolim P, Kusek JW, Pfeffer MA, Liu S, Eaton CB. Serum Calcification Propensity and Fetuin-A: Biomarkers of Cardiovascular Disease in Kidney Transplant Recipients. *Am J Nephrol.* 2018;48(1):21-31. doi: 10.1159/000491025. Epub 2018 Jul 11. PMID: 29996127
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  110. Malhotra R, Katz R, Weiner DE, Levey AS, Cheung AK, **Bostom AG**, Ix JH. Blood Pressure, Chronic Kidney Disease Progression, and Kidney Allograft Failure in Kidney Transplant Recipients: A Secondary Analysis of the FAVORIT Trial. *Am J Hypertens.* 2019 Aug 14;32(9):816-823. doi:

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113. Matías-García PR, Ward-Caviness CK, Raffield LM, Gao X, Zhang Y, Wilson R, Gao X, Nano J, **Bostom A**, Colicino E, Correa A, Coull B, Eaton C, Hou L, Just AC, Kunze S, Lange L, Lange E, Lin X, Liu S, Nwanaji-Enwerem JC, Reiner A, Shen J, Schöttker B, Vokonas P, Zheng Y, Young B, Schwartz J, Horvath S, Lu A, Whitsel EA, Koenig W, Adamski J, Winkelmann J, Brenner H, Baccarelli AA, Gieger C, Peters A, Franceschini N, Waldenberger M. DNAm-based signatures of accelerated aging and mortality in blood are associated with low renal function. *Clin Epigenetics*. 2021 Jun 2;13(1):121. doi: 10.1186/s13148-021-01082-w.PMID: 34078457
114. Bostom AG, Kenyon T, Eaton CB. Covid-19 positive test cycle threshold trends predict covid-19 mortality in Rhode Island. 2021.01.26.21250557; medRxiv doi: <https://www.medrxiv.org/content/10.1101/2021.01.26.21250557v1>

PubMed link for 117 total publications: <http://bit.ly/2t2yPb8>

#### **EDITORIALS, JOURNAL REVIEWS, AND BOOK CHAPTERS (invited, and not peer reviewed)**

1. **Bostom AG**, Dworkin LD. Cystatin C measurement: improved detection of mild decrements in glomerular filtration rate versus creatinine-based estimates? *Am J Kid Dis* 2000; 36: 205-207.
2. **Bostom AG**, Gohh RY, Morrissey P. Hyperhomocysteinemia in chronic renal transplant recipients. *Graft* 2000; 3: 197-204.
3. Culleton BF, **Bostom AG**. Hyperhomocysteinemia in chronic renal disease. Chapter 9, pages 211-228. In: Cardiovascular disease in end-stage renal failure. Loscalzo J, London GM, Editors. Oxford University Press, 2000.
4. **Bostom AG**, Brown RS Jr, Chavers BM, Coffman TM, Cosio FG, Culver K, Curtis JJ, Danovitch GM, Everson GT, First MR, Garvey C, Grimm R, Hertz MI, Hricik DE, Hunsicker LG, Ibrahim H, Kasiske BL, Kennedy M, Klag M, Knatterud ME, Kobashigawa J, Lake JR, Light JA, Matas AJ, McDiarmid SV, Miller LW, Payne WD, Rosenson R, Sutherland DE, Tejani A, Textor S, Valentine HA, Wiesner RH. Prevention of post-transplant cardiovascular disease--report and recommendations of an ad hoc group. *Am J Transplant* 2002 July; 2(6):491-500.

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5. Friedman AN, **Bostom AG**. Hyperhomocysteinemia in Renal Disease. Chpt. 270. In: Harrison's Online, edited by Braunwald E, Fauci AS, Isselbacher KJ, Kasper dL, Hauser SL, Longo DL, Jameson JL, McGraw-Hill, 2001.

### OTHER PUBLICATIONS

1. **Bostom AG**. Abnormalities of lipoprotein metabolism in the nephrotic syndrome. *N Engl J Med* 1991; 324:697-8.
2. Breslow J, et al. N-Acetylcysteine and lipoprotein (a). *Lancet* 1992;339:126-7.
3. **Bostom AG**, et al. Serum lipoprotein (a) as a risk factor for extracranial acrotid artery atherosclerosis. *Mayo Clin Proc* 1992;67:303-4.
4. MacLean DB, et al. Postmenopausal estrogen therapy and cardiovascular disease. *N Engl J Med* 1992; 326:707-8.
5. **Bostom AG**, et al. Microalbuminuria, lipoproteins, and diabetic contro. *Ann Intern Med* 1993;118:312.
6. **Bostom AG**. Lipoprotein(a) risk in women and efficacy of ascorbate. *JAMA* 1994; 272:1169.
7. Craig WY, et al. Lipoprotein(a) concentration and risk of atherothrombotic disease. *JAMA* 1995;274:1198-9.
8. **Bostom AG**. Folic Acid fortification of food. *JAMA* 1996;275:681.
9. **Bostom AG**. Methionine loading, vitamin B6 status, and premature thromboembolic disease. *Ann Intern Med* 1996;125:419-20.
10. **Bostom AG**. Adjunctive drug therapy for acute myocardial infarction. *N Engl J Med* 1997;336:1455.
11. **Bostom AG**. Effects of fenofibrate and gemfibrozil on plasma homocysteine. *Lancet* 2001;358(9295);1811-2.
12. **Bostom AG**. Cost-effectiveness of homocysteine-lowering therapy to prevent coronary heart disease. *JAMA* 2002;287(2):190;discussion 191-2.
13. **Bostom AG, Sharaf B**. B vitamins and restenosis after coronary angioplasty. *N Engl J Med* 2002;346(14):1093-5.

### ABSTRACTS

1. Brosnan JT, Hall B, Selhub J, Nadeau MR, **Bostom AG**. Renal metabolism of homocysteine in vivo. *Biochem Soc Trans* 1995;23:470S.

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2. **Bostom AG**, Thorpe K, Beecroft ML, Nadeau MR, Jacques PF, Selhub J, Rosenberg IH, Churchill DN. Lack of association between serum total homocysteine levels and non-fatal myocardial infarction prevalence in a large peritoneal dialysis inception cohort. *Can J Cardiol* 1997;13 (Suppl B):314B (Abstract #1147).
3. Friedman AN, **Bostom AG**, Selhub J, Levey AS, Rosenberg IH, Pierratos A. Nocturnal Versus Standard Hemodialysis and Plasma Total Homocysteine Levels. *Am Soc. of Nephrology annual meeting*, 2001. Volume 12, p. 356.
4. Friedman AN, Hunsicker L, Selhub J, **Bostom AG**. "Correlates of Plasma C-Reactive Protein Levels in Patients with Type 2 Diabetes Mellitus and Nephropathy". *Am Soc. of Nephrology*, 2002, abstract #787, p. 630A.
5. **Bostom A**, Friedman A, Hunsicker L, Jacques P, Selhub J. A Prospective Study of C-Reactive Protein and Pooled Cardiovascular Disease Outcomes in the Irbesartan Type 2 Diabetic Nephropathy Trial Cohort. July, 2003.
6. **Bostom A**, Friedman A, Hunsicker L, Jacques P, Selhub J. A Prospective Study of Total Homocysteine and Pooled Cardiovascular Disease Outcomes in the Irbesartan Type 2 Diabetic Nephropathy Trial Cohort. July, 2003.
7. **Bostom A**, Shemin D, Steffes M; McKenney J, HuS. Low Dose (500 mg) once daily was matrix extended-release niacin lowers serum phosphorus and raises HDL cholesterol concentrations in hemodialysis patients. *Am Soc of Nephrology annual meeting 2009*. PO#1860.
8. **Bostom A**, Carpenter M, Kusek J, Hunsicker L, Jacques P, Levey A, Pfeiffer M, Selhub J. Homocysteine lowering in chronic stable renal transplant recipients: the FAVORIT trial. *Am Soc of Nephrology annual meeting 2009*. LB#001.
9. Maccubbin D, Tipping D, Kuznetsova O, Hanlon W, **Bostom A**. Extended-release niacin/laropiprant lowers serum phosphorus concentrations in dyslipidemic patients. *Am College of Cardiology annual meeting 2010*.
10. **Bostom A**. Hypophosphatemic Effect of Niacin in Patients with Type 2 Diabetes: A Randomized Trial". *Am Soc of Nephrology annual meeting 2010*.

#### **INVITED PRESENTATIONS**

1. First International Conference on Homocysteine Metabolism, Dromoland Castle, Ireland, July 1-5, 1995. "Net Uptake of Homocysteine by the Rat Kidney In Vivo", July 3, 1995.
2. "Hyperhomocysteinemia in End-Stage Renal Disease." McMaster University, Hamilton, Ontario, Canada, Renal Grand Rounds, October 30, 1995.
3. "Hyperhomocysteinemia and Vascular Disease." Cardiology Grand Rounds. Stanford University, Palo Alto, CA, March 7, 1996.

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4. “Homocysteine and Vascular Disease.” 11th National Conference on Thrombosis and Hemostasis, November 8, 1996.
5. “Homocysteine and Vascular Disease in ESRD (End-Stage Renal Disease).” International Society of Nephrology, 13th World Congress, Sydney, Australia, May 26, 1997.
6. “Prospective Studies of Elevated Plasma Lipoprotein (a) and Coronary Heart Disease: A Critical Review.” University of Oslo, Institute of Medical Genetics, Special Two-Day International Seminar on Lipoprotein (a), May 30, 1997.
7. “Clinical Significance of Hyperhomocysteinemia.” Seventh Annual Symposium on Peripheral Vascular Disease, Newport, RI, August 15, 1997.
8. “B-Vitamins, Homocysteine, and Vascular Disease.” Cardiovascular Nutraceuticals Conference, Washington, DC, October 14-15, 1997.
9. Two-Day Visiting Professorship at The University of Iowa Medical Center, October 16-17, 1997.
- 9a. “Homocysteine and Vascular Disease.” Medicine Grand Rounds, October 16, 1997.
- 9b. “Hyperhomocysteinemia in End Stage Renal Disease: Prevalence, Etiology, and Potential Relationship to Arteriosclerotic Outcomes.” Cardiovascular Center Research Seminar, October 17, 1997.
10. “Identification and Treatment of Hyperhomocysteinemia.” Medicine Grand Rounds, Rhode Island Hospital, October 28, 1997
11. “Homocysteine-Lowering Trial in Renal Transplant Recipients.” 1997 Annual Meeting of the American Society of Nephrology, San Antonio, Texas, November 3, 1997.
12. “Management of Severe Hyperlipidemia with Presentation of Cases.” Cardiology Grand Rounds, Memorial Hospital of Rhode Island, December 2, 1997.
- 13a. “Serum Total Homocysteine Levels Predict All Cause and Cardiovascular Disease Mortality in Elderly Framingham Men and Women.” 38th Annual Conference on Cardiovascular Disease Epidemiology and Prevention, Sante Fe, NM, March 18-21, 1998.
- 13b. “Homocysteine - Another Cholesterol?,” 38th Annual Conference on Cardiovascular Disease Epidemiology and Prevention, Sante Fe, NM, March 18-21, 1998.
14. Second International Conference on Homocysteine Metabolism, Nijmegen, The Netherlands, April 26-29, 1998. “Homocysteine in Renal Disease.” Chairperson, Symposium, April 29, 1998.
15. “Homocysteine and Vascular Disease.” Medical Grand Rounds, Memorial Hospital of Rhode Island, May 13, 1998.
16. “Excess Prevalence of Hyperhomocysteinemia Among Chronic Renal Disease Patients Persists in the Era of Folic Acid Fortified Cereal Grain Flour.” 1<sup>st</sup> Homocysteinemia and Atherosclerosis RFA Grantees Meeting, National, Heart, Lung, and Blood Institute, Bethesda, MD, October 27, 1999.

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- 17a. “Mild Fasting Hyperhomocysteinemia in the Era of Folic Acid Fortification of Cereal Grain Flour: Comparison of Renal Transplant and Coronary Artery Disease Patients.” 71<sup>st</sup> Scientific Sessions, American Heart Association, Dallas, TX, November 9, 1998.
- 17b. “Non-Fasting Plasma Total Homocysteine Levels and Stroke Occurrence in Elderly Framingham Women and Men: A Prospective Study.” 71<sup>st</sup> Scientific Sessions, American Heart Association, Dallas, TX, November 10, 1998.
18. “Problem Cases in Hyperlipidemia.” Cardiology Grand Rounds, Memorial Hospital of Rhode Island, November 17, 1998.
19. “Homocysteine and Vascular Disease.” Cardiology Grand Rounds, Hartford Hospital, Hartford, CT, January 5, 1999.
20. “Homocysteine, Arteriosclerosis, and the ‘Reverse Causality’ Hypothesis: Ignorance of Renal Function is Not Bliss.” 39<sup>th</sup> Annual conference on Cardiovascular Disease Epidemiology and Prevention, Omni Rosen Hotel, Orlando, Florida, March 26, 1999.
21. “Homocysteine and Vascular Disease.” Medicine Grand Rounds, University of Nebraska Medical Center, Omaha, Nebraska, April 23, 1999.
- 22a. “Homocysteine Levels in Renal Transplantation: Determinants in the Era of Folic Acid Fortified Flour.” 18<sup>th</sup> Annual Meeting of the American Society of Transplantation, Chicago, IL, May 17, 1999.
- 22b. “Excess Prevalence of Mild Fasting Hyperhomocysteinemia Among Renal Transplant Versus Coronary Artery Disease Patients in the Era of Folic Acid Fortified Cereal Grain Flour.” 18<sup>th</sup> Annual Meeting of the American Society of Transplantation, Chicago, IL, May 17, 1999
23. “Homocysteine and Vascular Disease.” 51<sup>st</sup> Annual Meeting of the American Association of Clinical Chemistry, New Orleans, LA, July 27, 1999.
24. “Addressing Efficacy, Safety, and Outcomes: B-Vitamin Treatment of Hyperhomocysteinemia for Primary and Secondary Cardiovascular Disease Prevention.”, 82<sup>nd</sup> Annual Meeting of the American Dietetics Association, Atlanta, GA, October 17, 1999.
- 25a. “Homocysteine: ‘Expensive Creatinine’, or Important, Modifiable Risk Factor for Arteriosclerotic Outcomes in Renal Transplant Recipients?”, 32<sup>nd</sup> Annual Meeting of the American Society of Nephrology, Miami, FL, November 6, 1999.
- 25b. “Enhanced Reduction of Fasting Total Homocysteine Levels with Supraphysiological Versus Standard Multivitamin Dose Folic Acid Supplementation in Renal Transplant Recipients” 32<sup>nd</sup> Annual Meeting of the American Society of Nephrology, Miami, FL, November 6, 1999.
- 25c. “B12, But Not Folate Status, is an Independent Determinant of Fasting Total Homocysteine Levels in Renal Transplant Recipients 50 Years of Age and Older.” 32<sup>nd</sup> Annual Meeting of the American Society of Nephrology, Miami, FL, November 6, 1999.

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26. “Enhanced Reduction of Fasting Total Homocysteine Levels with Supraphysiological Versus Standard US Recommended Daily Allowance Dose Folic Acid Supplementation in Renal Transplant Recipients” 72<sup>nd</sup> Scientific Sessions, American Heart Association, Atlanta, GA, November 7, 1999.
27. “Homocysteine: ‘Expensive Creatinine’, or Important, Modifiable Risk Factor for Arteriosclerotic Outcomes in Renal Transplant Recipients?”, 2<sup>nd</sup> Homocysteinemia and Atherosclerosis RFA Grantees Meeting, National, Heart, Lung, and Blood Institute, Bethesda, MD, November 30, 1999 (oral presentation).
28. “Enhanced Reduction of Fasting Total Homocysteine Levels with Supraphysiological Versus Standard Multivitamin Dose Folic Acid Supplementation in Renal Transplant Recipients.”, 49<sup>th</sup> Annual Scientific Session, American College of Cardiology, Anaheim, CA, March 12, 2000 (poster presentation).
29. Division of Nephrology Grand Rounds, St. Joseph's Health Centre, "Homocysteine: Expensive Creatinine, or Important Modifiable Risk Factor for CVD Outcomes in Chronic Renal Disease?", Visiting Professor, Division of Nephrology, University of Western Ontario, Hamilton, Ontario, September 19, 2000 (oral presentation).
30. Clinical Pharmacology Grand Rounds, Robarts Research Institute, "Screening and Treatment Guidelines for Hyperhomocysteinemia: The Case for Watchful Waiting", Visiting Professor, Division of Nephrology, University of Western Ontario, Hamilton, Ontario, September 19, 2000 (oral presentation).
31. “Homocysteine and Vascular Disease”, Philadelphia Endocrine Society, Philadelphia, PA, October 11, 2000 (oral presentation).
32. “Reduced Foliates Are an Expensive Treatment for Hyperhomocysteinemia in Hemodialysis Patients with No Greater Efficacy Than Folic Acid.”, 33<sup>rd</sup> American Society of Nephrology Meetings, Toronto, Canada, October 13, 2000 (oral presentation).
33. “Chronic Renal Transplantation: A Model for the Hyperhomocysteinemia of Renal Insufficiency.”, 33<sup>rd</sup> American Society of Nephrology Meetings, Toronto, Canada, October 13, 2000 (poster presentation).
34. “Proteinuria and Total Plasma Homocysteine Levels in Chronic Renal Disease: Critical Impact of True Glomerular Filtration Rate.”, 33<sup>rd</sup> American Society of Nephrology Meetings, Toronto, Canada, October 13, 2000 (poster presentation).
35. “Cyclosporine Use is Not Independently Associated with Increased Plasma Total Homocysteine Levels in Austrian or United States Chronic Renal Transplant Recipients.”, 33<sup>rd</sup> American Society of Nephrology Meetings, Toronto, Canada, October 13, 2000 (poster presentation).
36. Renal Division Grand Rounds, “Total Homocysteine Lowering for the Potential Reduction of Arteriosclerotic Outcomes in Stable Renal Transplant Recipients: Rationale for a Multicenter Randomized Controlled Clinical Trial”, Boston University Medical Center, February 7, 2001 (oral presentation).

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37. “Controlled Comparison of L-Folinic Acid Versus Folic Acid for the Treatment of Hyperhomocysteinemia in Hemodialysis Patients.”, 41<sup>st</sup> Annual Conference on Cardiovascular Disease Epidemiology and Prevention, San Antonio, Texas, March 2, 2001 (poster presentation).
38. “Total Homocysteine Lowering Treatment Among Coronary Artery Disease Patients in the Era of Folic Acid Fortified Cereal Grain Flour.”, 41<sup>st</sup> Annual Conference on Cardiovascular Disease Epidemiology and Prevention, San Antonio, Texas, March 2, 2001 (poster presentation).
39. “Renal Insufficiency, Vitamin B12 Status, and Population Attributable Risk for Mild Hyperhomocysteinemia Among Coronary Artery Disease Patients in the Era of Folic Acid Fortified Cereal Grain Flour.”, 3<sup>rd</sup> Homocysteinemia and Atherosclerosis RFA Grantees Meeting, National, Heart, Lung, and Blood Institute, Bethesda, MD, March 6, 2001 (oral presentation).
40. “Total Homocysteine Lowering for the Potential Reduction of Arteriosclerotic Outcomes in Renal Transplant Recipients: Rationale for a Controlled Clinical Trial.”, Clinical Nephrology Meetings 2001, National Kidney Foundation, Orlando, Florida, April 21, 2001 (oral presentation).
41. "The Kidney and Homocysteine Metabolism", ASN/ISN World Congress of Nephrology, San Francisco, CA, October 10-17, 2001.
42. "Management of dyslipidemia in Chronic Renal Disease", Renal Grand Rounds, March 22, 2002.
43. "Measurement Parameters and Methodologies in Chronic Renal Disease, ASN Renal Week, Philadelphia, PA, November 1, 2002 (poster presentation).
44. "Folic Acid for Vascular Outcome Reduction in Transplantation (FAVORIT)", 1<sup>st</sup> Semi-Annual Renal Clinical Trails Network Consortium, Bethesda, MD, February 4-5, 2003.
45. Workshop on Cardiovascular Disease in Chronic Kidney Disease. Bethesda, MD, March 10-11, 2003.
46. "Independent Association Between C-Reactive Protein and Circulation vitamin B6, Body Mass Index, and Serum Creatinine in Type 2 Diabetic Nephropathy. Brown University, Dept. of Medicine, Ninth Annual Research Forum, Providence, RI, June 10, 2003 (poster presentation).
47. "Kidney & Other Organs in Metabolic Regulation of tHcy", Fourth International Conference on Homocysteine Metabolism, Basel, Switzerland, June 29-July 3, 2003.
48. “Homocysteine and Cardiovascular Disease Outcomes in Chronic Renal disease”, American Society of Nephrology Annual Meeting, Philadelphia, PA, November 10, 2005.
49. “FAVORIT Things: Rationale, Design, and Baseline Characteristics of the Folic Acid for Vascular Outcome Reduction in Transplantation Clinical Trial”, Rockefeller University, New York, New York, September 10, 2008.

**NIH SPECIAL EMPHASIS PANEL**



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1. "The African American Study of Kidney Disease and Hypertension (AASK): Cohort Study" (RFA) DK-03--03-500, NIDDK, Bethesda, MD, April 2, 2003.

## GRANTS

- 1/93-4/93 Principal Investigator  
Effect of high dose ascorbate (Vitamin C) on plasma lipoprotein (a) and homocysteine concentrations in patients with coronary heart disease  
Roche Vitamin Division \$5,000
- 1/95-6/95 Principal Investigator  
Effect of high dose B-vitamin supplementation on plasma total homocysteine levels in maintenance dialysis patients  
Roche Vitamin Division \$5,000
- 1/96-12/97 Principal Investigator  
Effect of B-vitamin supplementation on post-methionine load homocysteine concentrations in patients with coronary heart disease  
Roche Vitamin Division \$20,000
- 6/95-6/96 Principal Investigator  
Treatment of hyperhomocysteinemia in maintenance dialysis patients  
National Kidney Foundation, Massachusetts/Rhode Island Affiliate \$10,000
- 4/96-4/97 Principal Investigator  
Homocysteine and non-fatal myocardial infarction prevalence in a large peritoneal dialysis inception cohort  
R&D Laboratories \$16,000  
Baxter Corporation-Renal Division \$12,000
- 1/97-1/98 Principal Investigator  
Homocysteine and vascular disease incidence in CANUSA  
Grant-in Aid, American Heart Association (National) \$53,845
- 4/97-3/99 Co-Investigator for RO1-HL56908-01A1  
Homocysteine lowering trial in vascular disease patients  
National Heart, Lung, and Blood Institute \$692,616  
Principal Investigator for subcontract related to this grant to:  
Memorial Hospital of Rhode Island site, total subcontract \$268,541 over two years
- 6/98-6/99 Co-Investigator  
Treatment of hyperhomocysteinemia in renal transplant recipients  
National Kidney Foundation, Massachusetts/Rhode Island Affiliate \$15,000
- 9/99-9/00 Principal Investigator

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Treatment of Hyperhomocysteinemia in Hemodialysis Patients  
Surdna Fellowship, Brown University \$15,000

- 4/01-3/03 Principal Investigator for RO1- HL67695-01  
Serum Total Homocysteine and C-Reactive Protein as Predictors of Arteriosclerotic Outcomes in  
The Irbesartan Type 2 Diabetic Nephropathy Trial (IDNT)  
National Heart, Lung, and Blood Institute \$509,800, over 2-years.
- 7/01-1/12 Principal Investigator for RO1 DK56846-02  
A randomized, controlled trial of total homocysteine lowering to reduce cardiovascular disease  
outcomes in stable renal transplant recipients.  
National Institute of Diabetes, Digestive, and Kidney Diseases, \$15,300,000 over 5-years.
- 8/10-7/11 Principal Investigator  
A randomized, placebo-controlled, pilot study of the effects of wax matrix extended-release  
niacin on laboratory measures of calcium-phosphorus homeostasis and bone formation in stage  
3-4 kidney disease patients.  
National Center for Research Resources -COBRE Chen Pilot Project 1 year \$50,000
- 4/14-3/17 Consultant Bostom; Principal Investigator-Ix  
RO1-DK101720) “The Effects of Niacin on Mineral Metabolism in Chronic Kidney Disease”

### **ACTIVE**

Prevent Cancer Foundation Grant 1/14/2016-1/14/2018  
Principal Investigator-Bostom  
“Nicotinamide for actinic keratosis prevention in kidney transplant recipients”

NIDDK Awarded 2/9/2017  
1 X01 DK113962, NIDDK Repository request 21306  
Serum Magnesium, Cardio-Renal Events, and Total Mortality in the FAVORIT Trial Cohort  
(Modified request to measure in addition to serum magnesium, serum calcification propensity, fetuin A,  
uromodulin, glycated albumin, and fructosamine, approved by NIDDK March 15, 2017)

### **UNIVERSITY TEACHING ROLES**

#### **QUEENS COLLEGE GRADUATE SCHOOL**

1986-1988 Taught Cardiac Rehabilitation didactic course to MS in Exercise Physiology candidates, and  
oversaw practicum field experiences

### **HOSPITAL TEACHING ROLES**

Andrew G. Bostom, M.D., M.S. – October 27, 2021

- 1993-present Instructing Internal Medicine Residents and Attending Physicians, as well as Cardiology Fellows and Attending Cardiologists, in cardiovascular disease risk factor identification and management, Rhode Island Hospital
- 1997-present Instructing Internal Medicine and Family Medicine Residents and Attending Physicians, as well as Cardiology Fellows and Attending Cardiologists, in cardiovascular disease risk factor identification and management Memorial Hospital of Rhode Island
- 1998-present Lecturer for second-year medical students, Brown University School of Medicine, for Pharmacology Course: “Lipid-Lowering Drugs.”

**MANUSCRIPT REVIEWER**

American Journal of Cardiology	Journal of the American Medical Association
Journal of the American Society of Nephrology	Kidney International
New England Journal of Medicine	American Journal of Clinical Nutrition
Atherosclerosis	Circulation
Journal of the American College of Nutrition	Pediatrics
Nephron	Nephrology, Dialysis, and Transplantation